

Influence of Perfusion and Ventilation Scans on Therapeutic Decision Making and Outcome in Cases of Possible Embolism

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We examined the influence of perfusion (Q) and ventilation (V) scans on therapeutic decision making and outcome among 229 patients referred for lung scans because embolism was suggested and found that specific V/Q scan patterns strongly influenced postscan decisions regarding initiation, maintenance or cessation of heparin therapy. These therapeutic decisions bore a relationship to outcome (recurrences and death) and disclosed decision-making deficits that need remedy by future investigational and educational efforts.

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In patients suspected of having pulmonary embolism, perfusion (Q) and ventilation (V) lung scans are frequently done. However, there has been no systematic evaluation of how scan results influence therapeutic decision making or relate to patient outcomes.

We have examined these issues in a retrospective analysis of 229 cases of patients with possible embolism referred to the University of California, San Diego (UCSD), Nuclear Medicine Department for perfusion scans during a one-year period. The results indicate that, at our institution, specific scan patterns do influence decision making and also relate to patient outcome in terms of thromboembolic recurrence and death due to embolism.

Patients and Methods

The hospital records of all patients referred for perfusion lung scanning to rule out pulmonary embolism at the UCSD Medical Center during 1979 were reviewed. There were 236 such patients, 7 of whom had a pulmonary angiogram and were excluded from further analysis. In the remaining 229 patients, admission diagnoses, risk factors, signs and symptoms and laboratory data that antedated the request for lung scanning were recorded. All perfusion scans included four views (anterior, posterior and both laterals) and most included six views (both posterior obliques). Patients received about 2 mCi of macroaggregates labeled with technetium

99m, and imaging was done with a large-field-of-view gamma camera (Picker 4/15) centered over the 140 keV photopeak and using a 20% window with a high-resolution, low-energy, parallel-hole collimator.

Physicians in nuclear medicine decided, after review of the Q scan and a chest x-ray film, whether or not a ventilation scan was warranted. If warranted, ventilation scans were done immediately after perfusion imaging.¹ They consisted of a wash-in phase during tidal breathing, an equilibrium image and a seven-minute wash-out phase. The same camera was used, employing a 15% window centered on the 81-keV photopeak.

Patients were placed into five subgroups according to the original perfusion scan report (Table 1): (1) perfusion scan showing no abnormalities, (2) Q scan defect(s) limited to area(s) of chest film abnormality, (3) single subsegmental defects, (4) multiple subsegmental defects and (5) single or multiple segmental (or larger) defects. If more than one type of defect was present, the scan was categorized according to the largest defect reported. A chest x-ray film was taken in all patients within 24 hours of—usually just before—the lung scan. All lung scans and chest films were also reviewed independently by two of us (A.J.M. and M.S.K.). Disagreements with the original report, or between reviewers, occurred in only four instances. However, all scan results were classified according to the original report provided. In all, 51 of the

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perfusion scans (22%) showed no abnormalities. About half of these patients had been started on heparin therapy before the scan; none received heparin after the scan unless venous thrombosis was diagnosed. The very low rates of recurrence and death due to embolism in this group have been reported in detail elsewhere,² and this group will not be further discussed.

Patient follow-up in the remaining groups was achieved by reviewing all inpatient and outpatient records subsequent to the index scan. All patients were followed by faculty or house officers of the Department of Medicine. If records disclosed no subsequent patient visit for at least a month after discharge, an effort was made to contact the patient directly. If this effort failed, the patient was placed in the "no follow-up" category.

Three therapeutic decisions made by the attending physicians during the index admission were examined: the prescan decision regarding anticoagulant use, the postscan decision and the decision to maintain anticoagulant therapy following discharge.

With respect to outcome, we analyzed the frequency of thromboembolic recurrence and of death due to embolism during the index admission and during follow-up. The criteria used to define both recurrence of venous thrombosis or embolism and death due to embolism were as follows:

- **Thromboembolic recurrence.** This was diagnosed if, after the initial lung scan, either of the following criteria was met: the results of a specific diagnostic test indicated recurrence of pulmonary embolism (lung scan or pulmonary angiogram) or deep venous thrombosis (venogram, impedance plethysmography or radiofibrinogen leg scan); a thromboembolic recurrence was entertained clinically by the attending physicians and led to extension of the index admission, a subsequent hospital admission or initiation of anticoagulant therapy. Unless a definitive study ruled out a recurrence, all such patients were counted as having a recurrence.

- **Death due to embolism.** Death was attributed to embolism when (1) autopsy was done and disclosed pulmonary emboli, or (2) autopsy was not done, but a clinical diagnosis of embolism was considered the cause of death, or anticoagulant therapy was applied during final admission because of a diagnosis of embolism or a scan or pulmonary angiogram documented embolic recurrence. Statistical analyses were done using the Fisher's exact test.

Results

The distribution of the 229 patients among the lung perfusion scanning subgroups is shown in Table 1. Ventilation scans were done in 120 instances (52%) (Table 1). Those 109 patients in whom nuclear medicine physicians decided not to do a ventilation study fell into one of four categories: (1) a perfusion scan showed no abnormalities (50), (2) their perfusion scan defects exclusively matched chest film abnormalities (25), (3) perfusion scan defects were considered too small (subsegmental) to resolve with xenon 133 (29) and (4) those with segmental or larger defects who were too ill to cooperate for a ventilation study (5).

Patient Risk Profiles

The patient risk profiles in the three major scan subgroups analyzed are presented in Table 2. Patients in each subgroup had significant risk factors for thromboembolism.³⁻¹² No sta-

tistically significant differences ($P < .05$) were noted among the groups for coexistent disease states, age, sex or race, presenting symptoms, signs or arterial blood gas analysis (not listed). This was true whether patients with segmental or larger defects were analyzed as one group or, as in Table 2, according to ventilation match or mismatch.

Decision Making and Outcome in Subgroups

Single subsegmental defects. Only ten patients had single subsegmental defects. Ventilation scans were done in six and five showed V/Q match. In three patients, heparin had been initiated before the ventilation scan and, in all, therapy was discontinued after the scan. Thus, no patient received heparin after a scan. No recurrences or deaths occurred in this group of patients during admission or during an average of seven months of follow-up.

Perfusion scan defects matched by chest film findings. Of the 31 patients in this category, 6 had a ventilation scan. In all, the ventilation defect(s) "matched" perfusion defect(s).

Seven patients had been started on heparin therapy before the scan (Table 3). After the scan, heparin therapy was continued in three of these and initiated in none. Thus, 28 patients received no anticoagulants following lung scanning.

During the index admission, there were no recurrences of thromboembolism. Four patients died during admission, none due to embolism according to the criteria described;

TABLE 1.—Perfusion Scans Classified by Category, Showing Scan Results in Terms of Ventilation/Perfusion (V/Q) Matching

Perfusion (Q) Scan Pattern*	Number	Ventilation (V) Scan Done Number (Percent)	V/Q Match Number	V/Q Mismatch Number (Percent)
Q normal	51	1 (2)	1	0 (0)
Q single SS	10	6 (60)	5	1 (17)
Q matches CXR	31	6 (19)	6	0 (0)
Q multiple SS	85	60 (71)	60	0 (0)
Q segmental	52	47 (90)	17	30 (64)
TOTAL	229	120 (52)	89	31

*Categories: Q normal = no abnormalities found on perfusion scan; Q single SS = a subsegmental defect found on perfusion scan; Q matches CXR = defect(s) on perfusion scan limited to area(s) of chest roentgenographic abnormality; Q multiple SS = subsegmental defects found on perfusion scan; Q segmental = segmental or larger defect(s).

TABLE 2.—Patient Profiles in Three Perfusion Scan (Q) Subgroups*

Patients	Q Matches CXR		Q Multiple Subsegmental		Q segmental V Match-V Mismatch	
	Number	Percent	Number	Percent	Number	Percent
Number	31		85		17	30
Average age, years	49		59		49.6	50.9
M/F	1.1/1		1.2/1		1.4/1	1.7/1
Risk Factors†	Number (Percent)		Number (Percent)		Number (Percent)	Number (Percent)
Smoker/COPD	11 (35)		62 (73)		12 (71)	18 (60)
Heart disease	13 (42)		42 (49)		5 (29)	8 (27)
Cancer	11 (35)		9 (11)		2 (12)	12 (40)
Immobilization	10 (32)		13 (15)		5 (29)	9 (30)
Postoperation	7 (23)		5 (6)		0 (0)	6 (20)
Hx DVT/PE	0 (0)		5 (6)		0 (0)	3 (10)
BCP/estrogens	2 (6)		2 (2)		3 (18)	4 (13)

*Defect(s) on perfusion scan matches region(s) of chest roentgenographic infiltrate; multiple subsegmental defects; or segmental defects with ventilation scan (V) match or mismatch.

†COPD = chronic obstructive pulmonary disease; Hx DVT/PE = history of deep venous thrombosis or pulmonary embolism, or both; BCP/estrogens = recent (within prior 3 months) use of oral birth control medication or estrogens.

none were receiving anticoagulants. Three patients were discharged on a regimen of long-term anticoagulation.

Of the 27 patients discharged, 24 (89%) were followed for an average of seven months (Table 4). One subsequent documented (by angiogram) embolic event occurred (4%). This patient had received heparin therapy for ten days during his index admission three months before. There were four deaths during follow-up; none was attributed to embolism; one patient was receiving anticoagulant therapy long-term.

Multiple subsegmental defects. There were 85 patients in this subgroup; 60 of these patients had a ventilation scan, all showing V/Q "match" (Table 1).

Heparin therapy had been started before the scan in 25 of these patients (29%) (Table 3). Following the lung scan, heparin therapy was discontinued in 11 patients and not initiated in any. Thus, only 14 patients (16%) were maintained on heparin therapy after the scan.

During the index admission, there was one recurrence, of pulmonary embolism, in a patient four days after completion of a ten-day course of heparin. He was re-treated and discharged. There were three deaths during admission; none was attributed to embolism; two of the patients were receiving heparin when death occurred. Postmortem examination in two, one receiving heparin and one not, disclosed no emboli.

Eighteen patients were discharged on long-term anticoagulant therapy, even though only 14 of these had been treated with heparin after a perfusion scan. The other four patients

were felt to have risk factors at discharge warranting such therapy.

Of the 82 patients discharged, 78 (95%) were followed for an average of eight months (Table 4). There were no recurrences. Five patients died during the follow-up period, three of whom were receiving anticoagulant therapy. None of the deaths was attributable to embolism.

Segmental defects. Of 52 patients whose perfusion scans showed at least one segmental or greater defect, 47 (90%) had a ventilation study (Table 1); 17 of these patients had V/Q mismatch.

Of the 17 with a segmental V/Q match pattern, 6 had been started on heparin therapy before the scan (Table 3). The heparin regimen was stopped after the scan in three of these patients and initiated in none. Thus, only three of these patients (18%) received postscan heparin. There were no recurrences during admission. There was one death, not attributed to pulmonary embolism, in a patient receiving heparin. No patient was discharged on a regimen of long-term anticoagulation. Of the 16 patients who survived the index admission, 15 (94%) were followed for a period averaging nine months (Table 4). There were no episodes of subsequent embolism. One died of nonembolic causes.

Among the 30 patients with a segmental V/Q mismatch pattern, 15 (50%) had been started on heparin therapy before the scan. This was continued in all after the scan. Of the remaining 15, heparin therapy was initiated after the perfusion scan in 13 and 2 were not treated because the emboli were septic. Thus, all patients except those with septic emboli received heparin after the scan.

There were three embolic recurrences during admission despite heparin treatment: two were documented by new segmental V/Q "mismatch" zones; one was a paradoxical embolus to the brachial artery in a patient with venous thrombosis and a patent foramen ovale. Three patients died during the index admission; all deaths were attributed to embolism; all were receiving heparin; one postmortem examination was done and documented emboli. In all, 16 patients (59%) were discharged on long-term anticoagulation. The 11 patients not given anticoagulant therapy after discharge included 2 who had septic emboli; the rationale for anticoagulant cessation in the other 9 was not apparent.

Of the 27 patients surviving the index admission, 26 (96%) were followed for a period averaging nine months. Six patients had thromboembolic recurrences: one with documented venous thrombosis, the five others with clinically diagnosed embolism. Of these six, three were receiving anticoagulants at recurrence and three were not. Four patients died during follow-up; three deaths were attributed to embolism. Two were receiving anticoagulant therapy at the time of death; in one, heparin therapy was withheld because of a hemorrhagic cerebrovascular accident. One postmortem examination was done and disclosed pulmonary emboli.

Of the five patients with segmental or larger perfusion defects who had no ventilation scan, three had been started on heparin therapy pre-scan. Two were maintained on heparin therapy after the scan. There were no recurrences during admission. One death, not attributed to embolism, occurred. No patient was discharged on anticoagulant therapy. The four surviving patients were followed an average of eight months. There were no recurrences or deaths.

TABLE 3.—Decisions and Events During Index Admission of Patients in Whom Defects Found on Perfusion Scan (Q) (N = 168)

Perfusion Scan Pattern*	Patients Number	Anticoagulation Therapy		Outcome		Discharged on Anticoag Regimen
		Heparin Prescan Number (Percent)	Heparin Postscan Number (Percent)	Recurrence Number	Deaths/PE/ Autopsy†	
Q matches CXR . . .	31	7 (23)	3 (10)	0	4/0/0	3/27
Q multiple SS . . .	85	25 (29)	14 (16)	1	3/0/2	18/82
Q segmental	52					
V match	17	6 (35)	3 (18)	0	1/0/0	0/16
V mismatch	30	15 (50)	28 (93)	3	3/3/1	16/27
No V	5	3 (60)	2 (40)	0	1/0/0	0/4

Anticoag = anticoagulation

*Q matches CXR = defect(s) on perfusion scan limited to area(s) of chest roentgenographic abnormalities; Q multiple SS = multiple subsegmental defects on perfusion scan; Q segmental = segmental or larger defects on perfusion scan, in which case a ventilation scan (V) was likely to have been done.

†Total No. deaths/No. attributed to pulmonary embolism/No. autopsies.

TABLE 4.—Outcome of Patients (N = 156) During Follow-up Period

Perfusion Scan Patterns*	Available† Number	Follow-up‡ Number (Percent)	Average Duration of Follow-up Months	Recurrence		Deaths/PE/ Autopsy§
				PE	DVT	
Q matches CXR . . .	27	24 (89)	7	1		4/0/0
Q multiple SS . . .	82	78 (95)	8	0	0	5/0/0
Q segmental	47					
V match	16	15 (94)	9	0	0	1/0/0
V mismatch	27	26 (96)	9	5	1	4/3/1
No V	4	4 (100)	8	0	0	0/0/0

PE = pulmonary embolism, DVT = deep venous thrombosis

*See Table 3 for explanation of perfusion scan (Q) and ventilation scan (V) pattern.

†No. surviving index admission.

‡Patients followed for one month or more.

§Total No. deaths/No. attributed to pulmonary embolism/No. autopsies.

The incidence of thromboembolic recurrence during admission and follow-up (Tables 3 and 4) was significantly higher among patients with segmental V/Q mismatch than among those with segmental V/Q match ($P < .05$), those with multiple subsegmental defects ($P < .001$) and those who had perfusion defects that matched the findings of the chest film ($P < .01$).

Venous studies. Among the scan groups analyzed in this report, in only 50/168 patients (30%) was the diagnosis of venous thrombosis pursued by impedance plethysmography, radiofibrinogen leg scanning or venography, or all of these (Table 5). The timing of leg studies vis-à-vis the lung scan was inconsistent; it ranged from a day or two before the scan to several days after the scan. When done, these tests appeared to influence therapeutic decision making. Of the three patients with perfusion defects matched to chest film defects who were maintained on heparin therapy after the scan and discharged on heparin therapy, all had positive leg studies. Of the 14 with multiple subsegmental defects maintained on heparin therapy after the scan, 8 had positive leg studies. Among those with segmental or larger perfusion scan defects mismatched by ventilation scan findings, leg studies were positive in 14 of the 16 discharged on a regimen of heparin.

The inconsistent timing and application of venous studies made more precise analyses of their influence on decisions or outcome impractical.

Discussion

Classical studies of diagnostic techniques for venous thromboembolism assess their sensitivity, specificity and reproducibility.¹³⁻²⁴ While such information is important, that was not our objective. Rather, we wished to assess the influence of lung scans on physician decision making and how these decisions conditioned patient outcome. Such assessments have not previously been reported.

Clearly, one cannot assess physician decision making if a protocol is in place that mandates such decisions. Furthermore, at the time of this study, there was no policy at our institution that mandated any of the decisions taken, nor was there an unstated, but generally accepted, philosophy. Our findings reflect the fact that there was nonuniformity in physician decision making regarding the following aspects of pa-

tient management: whether to do a ventilation scan, when heparin therapy should be initiated or discontinued, how such patients are best followed and how best to confirm the diagnosis of recurrence. Such nonuniformity is not condoned by the authors; it is merely reported. Despite the interpretive limitations that such variability imposes, our data indicate that perfusion and ventilation scan patterns do influence physician decision making and do relate to patient outcome.

The first therapeutic decision was whether to initiate heparin therapy on the basis of data available *before* the lung scan. Of the 168 patients, the decision was to initiate heparin therapy in 56 (33%).

The second decision was whether to maintain, initiate or discontinue administration of heparin after the scan. Among the 56 started on heparin therapy pre-scan, it was discontinued in 19 patients after the scan (34%). Considering that heparin therapy also was discontinued in virtually all of those in whom no abnormalities were found on a perfusion scan, it is clear that scans did influence decisions to discontinue heparin therapy. Furthermore, scan results also led to the initiation of heparin therapy in 13 patients who had not been given heparin before the scan.

Examination of the various scan subgroups indicates that these decision changes were not random but were conditioned by specific scan patterns. Among patients with perfusion scans showing defect(s) limited to area(s) of pulmonary infiltrates seen on chest film, heparin therapy was initiated in none and maintained in only three (all had venous thrombosis). The ventilation scan did not appear to influence these decisions. Likewise, among the 85 patients with a perfusion scan showing multiple subsegmental defects, heparin therapy was initiated after the scan in none and maintained in only 14 (8 with positive leg studies). Again, ventilation scans did not influence these decisions.

Among patients with segmental or larger perfusion defects, the ventilation scan did appear to influence postscan therapeutic decisions. Of the 17 with V/Q match, only three received heparin after the scan, one because of venous thrombosis. In sharp contrast, of the 30 patients with V/Q mismatch, 15 had heparin therapy continued and 13 had heparin therapy *initiated* following the scan. Indeed, the only two patients who did not receive heparin after the scan had septic emboli.

TABLE 5.—Venous Studies Done in 168 Patients With Perfusion Scan Defects*

Perfusion Scan Pattern†	#	Leg Studies Number Done / Number Pos	Postscan Heparin Therapy Number/Pos Leg	Discharged on Anticoag Regimen Number/Pos Leg
Q matches CXR . . .	31	10/6	3/3	3/3
Q multiple SS . . .	85	13/9	14/8	18/5
Q segmental	52			
V match	17	3/1	3/1	0
V mismatch . . .	30	21/14	28/14	16/14
No V	5	3/2	2/2	0
	168	50/32	50/28	37/22

Anticoag = anticoagulation

*Studies done were venography, impedance plethysmography or radiofibrinogen leg scan. Of 50 patients studied by one or more techniques, a positive (pos) result was found in 32. A majority of those continued on heparin post-scan and discharged on anticoagulants had positive leg studies.

†See Table 3 for explanation of perfusion (Q) and ventilation (V) scan patterns.

The next therapeutic decision—whether to maintain anticoagulant therapy after discharge—appeared to have been conditioned by the scan results (Tables 3 and 5), the detection of venous thrombosis and considerations of long-term thromboembolic risk. Of the 37 patients discharged on a regimen of anticoagulation, 24 (65%) had either a scan pattern showing segmental or larger perfusion defects with ventilation mismatch, test findings of venous thrombosis, or both. Why the other patients were continued on anticoagulant therapy is not evident from the record, nor did the record reflect why long-term anticoagulation was not given to some patients, such as nine who had segmental or larger perfusion defects and V/Q mismatch. It seems evident that our physicians do not use consistent criteria in making decisions about long-term anticoagulant therapy.

However, the data indicating that scan patterns did influence therapeutic decision making do not answer the critical question: were the decisions appropriate? Only analysis of patient outcome can address that question.

In this series, the incidence of recurrence and of death due to embolism were the two outcome criteria. Unfortunately, precise identification of these events is difficult, whether patients are studied prospectively or retrospectively, as was reflected in a recent prospective study.²³ Because episodes of embolism may be asymptomatic,²⁴ even the most rigorous clinical follow-up will detect only recurrences that induce symptoms. Without symptoms, patients will not seek attention nor will physicians intervene. Unfortunately, the alternative is a study design that would mandate frequent follow-up scans or pulmonary angiograms among asymptomatic patients over periods of months or years, a design that is difficult to justify. Similarly, with respect to death due to embolism, a 100% autopsy rate is certainly desirable, but not attainable. In this study, we dealt with these problems by defining recurrence and death due to embolism quite liberally. This may lead to inclusion of excessive recurrences and deaths, but such a "worst case" analysis seems most appropriate. Despite such limitations, the outcome data were instructive. During the index admission, the decision to provide heparin therapy to a minority of patients with scan patterns other than segmental or larger V/Q mismatch was associated with only one recurrence and no embolic deaths. We have previously reported similar results in patients in whom anticoagulant therapy was discontinued after a perfusion scan showed no abnormalities.¹ In contrast, recurrence and embolic deaths occurred during the index admission in some 10% of those with segmental or larger perfusion defects mismatched by ventilation scan.

Long-term follow-up provided similar results. Among the 121 patients followed in the groups other than those with segmental V/Q mismatch, there was only one recurrence (of pulmonary embolism) and no deaths attributable to embolism. Yet less than 20% of these patients received anticoagulant therapy after discharge. In contrast, of the 26 patients with V/Q mismatch followed after discharge, there were six recurrences and three deaths attributable to embolism (two while patients were receiving heparin).

Whereas these outcome data suggest that the decisions regarding heparin therapy were generally appropriate, they leave several important questions unresolved. For example, physicians apparently concluded that a perfusion scan showing multiple subsegmental defects excluded embolism

and, therefore, that anticoagulant therapy was not necessary. The acceptable outcome data do not validate such conclusions. The acceptable outcome could mean that, whereas an unknown percentage had small emboli, such small emboli, even if recurrent, do not cause significant symptoms or result in death. Indeed, the recent report by Hull and colleagues²³ favors the latter conclusion. Although their study differed substantially in design, they found that the pulmonary angiogram disclosed pulmonary emboli in 27% of patients with subsegmental defects and 17% with perfusion scan defects matched by chest film findings, versus 86% with segmental or larger defects mismatched by ventilation scan. Thus, clearly, emboli do occur in these other scan subgroups. However, the outcome data in the series of Hull and co-workers also reflect, as we found, a low risk of subsequent recurrences and deaths. Of their 139 patients with abnormalities seen on perfusion scans, most received no anticoagulant therapy because, by design, a combination of a negative pulmonary angiogram and a negative venogram led to withholding of therapy. An unknown number received anticoagulant therapy after discharge. Yet, these workers found only three documented recurrences during a one-year follow-up: one embolic event and two instances of venous thrombosis. The one embolic recurrence occurred in a patient with a segmental V/Q mismatch scan pattern. Further, of 25 deaths during follow-up, they attributed only 3 to embolism, 1 each in the segmental V/Q mismatch, subsegmental and chest film-matched groups.

Thus, while our report and that of Hull and co-workers cannot be directly compared, it is evident that the outcome data in both studies suggest that recurrence of, and death due to, embolism is quite uncommon among patients whose index scans show subsegmental or roentgenogram matching patterns. Further data bearing on this point are needed.

Likewise, the uniform decision to treat patients who had segmental or larger defects with V/Q mismatching does not indicate that such patients had emboli. That this type of V/Q mismatch is commonly associated with embolism is strongly suggested by other studies in which angiographic-scan comparisons have been made in selected patient groups.^{15-19,23} As noted, the report of Hull and associates places this figure at 86%. The high recurrence or death rates (or both) we observed add further weight to the diagnostic implications of this scan pattern.

Another question that is stimulated, rather than resolved, by our observations is why studies for venous thrombosis were used so inconsistently in making initial and discharge decisions regarding anticoagulant therapy. Impedance plethysmography, radiofibrinogen leg scans and Doppler and contrast venography are all readily available at our institution. Yet, these studies were done in only a minority of these patients, even though most emboli come from lower extremity venous thrombosis.²⁰⁻²⁵ Obviously, additional educational efforts are needed to emphasize the value of venous studies among patients in whom embolism is suggested.

REFERENCES

1. Kipper MS, Alazraki N: The feasibility of performing ¹³³Xe ventilation imaging following the perfusion study. *Radiology* 1982; 144:581-586
2. Kipper MS, Kortman KE, Ashburn WL, et al: Long-term follow-up of patients with suspected pulmonary embolism and a normal lung scan. *Chest* 1982; 82:411-415
3. Foster CS, Genton E, Henderson M, et al (Eds): The epidemiology of venous thrombosis. *Millbank Mem Fund Q* 1971; 50:1-292
4. Frantantoni J, Wessler S: Risk Factors and the Epidemiology of Venous Thromboembolism in Prophylactic Therapy of Deep Vein Thrombosis and Pulmonary Embo-

EMBOLISM

lism, US Dept of Health, Education, and Welfare publication No. (NIH) 76-866. Government Printing Office, 1976, pp 18-27

5. Browse NL: Prevention of venous thromboembolism. *N Engl J Med* 1972; 287:145-148

6. Clagett GP, Salzman EW: Prevention of venous thromboembolism in surgical patients. *N Engl J Med* 1974; 290:93-97

7. Predisposition to thrombosis (Editorial). *Lancet* 1974; 2:1430

8. Kakkar VV, Corrigan TP, Fossard DP: Prevention of fatal post-operative embolism by low-dose heparin: An international multicenter trial. *Lancet* 1975; 2:45-51

9. Maurer BJ, Wray R, Shillingford JP: Frequency of venous thrombosis after myocardial infarction. *Lancet* 1971; 2:1385-1388

10. Moser KM: State of the art: Pulmonary embolism. *Am Rev Respir Dis* 1976; 115:829-852

11. Sasahara AA, Sharma GV, Parisi AF: New developments in the detection and prevention of venous thromboembolism. *Am J Cardiol* 1979; 43:1214-1219

12. Warlow C, Ogston D, Douglas AS: Venous thromboembolism following strokes. *Lancet* 1972; 1:1305-1307

13. Wallace JM, Moser KM, Hartman MT, et al: Patterns of pulmonary perfusion scans in normal subjects study. *Am Rev Respir Dis* 1981; 124:480-483

14. Wallace JM, Moser KM, Hartman MT, et al: Patterns of pulmonary perfusion scans in normal subjects study—II. The prevalence of abnormal scans in young non-smokers. *Am Rev Respir Dis* 1982; 125:465-467

15. Alderson PO, Biello DR, Kahn AR, et al: Comparison of ^{133}Xe single-breath and washout imaging in the scintigraphic diagnosis of pulmonary embolism. *Radiology* 1980; 134:481-483

16. Alderson PO, Lec H, Summer N, et al: Comparison of ^{133}Xe washout and single-breath imaging for the detection of ventilation abnormalities. *J Nucl Med* 1979; 20:917-922

17. Biello DR, Matter GA, Osei-Wusu A, et al: Interpretation of indeterminate lung scintigrams. *Radiology* 1979; 133:189-194

18. McNeil BJ: Ventilation-perfusion studies and the diagnosis of pulmonary embolism: Concise communication. *J Nucl Med* 1980; 213:319-323

19. McNeil BJ: A diagnostic strategy using ventilation-perfusion studies in patients suspect for pulmonary embolism. *J Nucl Med* 1976; 17:613-615

20. Kakkar VV, Corrigan TP: Detection of deep venous thrombosis: Survey and current status. *Prog Cardiovasc Dis* 1974; 17:207-218

21. Hull R, Hirsh J, Sackett DL, et al: Cost effectiveness of clinical diagnosis, venography and noninvasive testing in patients with symptomatic deep venous thrombosis. *N Engl J Med* 1981; 304:1561-1567

22. Hull R, Hirsh J, Sackett DL, et al: Replacement of venography in suspected venous thrombosis by impedance plethysmography and ^{125}I -fibrinogen leg scanning: A less invasive approach. *Ann Intern Med* 1981; 94:12-16

23. Hull RD, Hirsh J, Carter CJ, et al: Pulmonary angiography, ventilation lung scanning and venography for clinically suspected pulmonary embolism with abnormal perfusion lung scan. *Ann Intern Med* 1983; 98:891-899

24. Moser KM, LeMoine JR: Is embolic risk conditioned by location of deep venous thrombosis? *Ann Intern Med* 1981; 94:439-444

25. Wheeler HB, O'Donnell JH, Anderson FA, et al: Occlusive impedance plethysmography: A diagnostic procedure for venous thrombosis and pulmonary embolism. *Prog Cardiovasc Dis* 1974; 17:199-206